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Post Neurosurgical Meningitis due to Colistin Heteroresistant *Acinetobacter baumannii*

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Introduction: Recently *Acinetobacter baumannii* isolates have emerged as a problematic infectious agent that causes meningitis in neurosurgical patients. Colistin has been used successfully for the treatment of *A. baumannii* meningitis but colistin resistant isolates have been reported worldwide.

Case Presentation: Two isolates of *A. baumannii* were cultured during a five-day period from cerebrospinal fluid (CSF) samples of a 20-year-old man with a gunshot trauma in the abdomen, which had exited from his back. Antimicrobial susceptibility tests of isolates were performed. Multiplex PCR was performed for detection of $bla_{\text{OXA-23-like}}$, $bla_{\text{OXA-24-like}}$ and $bla_{\text{OXA-58-like}}$ genes. Metallo-β-lactamase genes such as: bla_{VIM}, bla_{IMP}, bla_{SPM} and bla_{NDM} were sought by singleplex PCR. In order to evaluate the genetic relationship, two isolates were examined by the repetitive extragenic palindromic-polymerase chain reaction (REP_PCR) method.

Conclusions: E-test results showed that the isolates were sensitive to colistin and tigecycline with minimum inhibitory concentration of (MIC) 0.25 μg/mL and 1.5 μg/mL, respectively. Secondly the isolates were resistant to colistin with MIC > 256 μg/mL but remained sensitive to tigecycline with MIC 1.5 μg/mL. On the basis of the multiplex PCR, both of the isolates were positive for $bla_{\text{OXA-23-like}}$. Other investigated genes such as $bla_{\text{OXA-24-like}}$, $bla_{\text{OXA-58-like}}$, bla_{VIM}, bla_{IMP}, bla_{SPM} and bla_{NDM} were negative. REP-PCR results showed that two isolates were derived from a single strain and both were the same. The results of our study revealed that the firs isolate of *A. baumannii* was colistin heteroresistant and was changed to completely resistant during therapy. Diagnosis and treatment of *A. baumannii* meningitis is very important and to avoid treatment failure we suggest that all *A. baumannii* isolates obtained from CSF should be evaluated properly for colistin heteroresistance.

Keywords:Acinetobacter baumannii; Meningitis; Colistin; Neurosurgery

1. Introduction

Acinetobacter baumannii has become an important pathogen that causes nosocomial infections such as bloodstream and wound infections, ventilator-associated pneumonia and meningitis (1). Approximately 10% of Gram-negative and 4% of nosocomial meningitis occur as a result of *A. baumannii* infection (2). Neurosurgical patients with cerebrospinal fluid (CSF) leakage have a high risk of acquiring meningitis. *A. baumannii* is a serious pathogen that causes meningitis in these patients (3) and many authors worldwide have reported post neurosurgical meningitis due to *A. baumannii* (4). Since *A. baumannii* strains are multidrug resistant, nosocomial infections associated with neurosurgical procedures such as nosocomial meningitis caused by *A. baumannii* had a high rate of mortality (5, 6); two studies have reported a mortality rate ranging from 71.4% to 72.7%, respectively for *A. baumannii* meningitis in neurosurgical patients (5, 7).

In the recent years, as a result of excessive use of antibiotics, the epidemiology of post-neurosurgical meningitis may have changed, and because treatment options for multidrug resistant *A. baumannii* are limited, the rate of meningitis due to this bacterium will be increased (2). Colistin is the last resort for treatment of pan drug-resistant *A. baumannii*, but increased use of this antibiotic has led to progress of colistin-resistant strains (1). The first colistin heteroresistance in *A. baumannii* was described in 2006. This type of resistance may be the primary stage that upon contact with colistin, resistant subpopulations can proliferate (8). Colistin heteroresistance seriously warns that colistin should be used properly. If colistin is used improperly, resistance may develop and lead to treatment failure (9). We report a case of post-neurosurgical meningitis due to colistin heteroresistant *A. baumannii*, with some changes in antibiotic susceptibility profile during intra-treatment with colistin.